

# Work hours, sleepiness and the underlying mechanisms

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**SUMMARY** Severe subjective and physiological sleepiness occur in night work, afflict almost all individuals and are associated with a performance impairment severe enough to explain night-work accident data. The alertness deficit is caused by the displacement of work to the circadian phase which is least conducive to alert behaviour, by extension of the time spent awake and by the reduction of sleep length (due to circadian interference with sleep). Sleepiness will be extreme when the three causes are operative simultaneously. The three factors may be used quantitatively to predict sleepiness.

**KEYWORDS** accidents, shiftwork, sleepiness, work hours.

## INTRODUCTION

As evidenced in the papers in this issue by Dinges pp. 4–14, Horne and Reyner pp. 23–29, Samel *et al.* pp. 30–36, and others in this volume, irregular work hours increase the risk of accidents, apparently mediated by sleepiness and its related performance decrements. The present paper will review in more detail the evidence of sleepiness in irregular work hours, and discuss the mechanisms.

## PERCEIVED SLEEPINESS

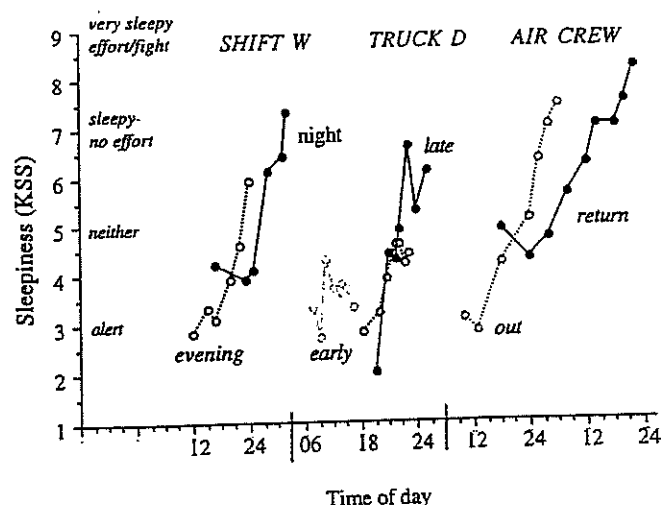
Sleepiness has been defined as a drive towards sleep (Dement and Carskadon 1982) and is traditionally expressed in subjective terms, although there are clearly pronounced behavioural and physiological expressions.

With respect to the prevalence of perceived sleepiness there is a wealth of questionnaire studies suggesting that the overwhelming majority of shift workers experience sleepiness in connection with night-shift work, whereas daywork is associated with no, or marginal, sleepiness (Wyatt and Mariott 1953; Thiis-Evensen 1957; Menzel 1962; Mott *et al.* 1965; Andersen 1970; Åkerstedt and Torsvall 1978; Verhaegen *et al.* 1981; Wagner and Garcia 1986; Gold *et al.* 1992; Paley and Tepas 1994). The studies by Verhaegen and Paley had an experimental design and showed that reported fatigue increased on entering and decreased on leaving shift

work. In many studies a majority of shift workers admit to having experienced involuntary sleep on the night shift, whereas this is rare on day-oriented shifts (Prokop and Prokop 1955; Kogi and Ohta 1975; Åkerstedt *et al.* 1983; Coleman and Dement 1986). Between 10 and 20% report falling asleep regularly during night work. Also work hours that include on-call duty will cause sleep/wake disturbances, as seen, for example, in physicians (Arnetz *et al.* 1990) or marine machine engineers (Torsvall and Åkerstedt 1988a). Finally, one should be aware of the possibility that individuals engaged in irregular work hours seem to underestimate the sleep/wake effects (Spelten *et al.* 1993).

When subjective rating scales have been administered repeatedly during the day and night, the results indicate moderate to high sleepiness during the night shift and no sleepiness at all during the day shifts (Fröberg *et al.* 1972; Folkard *et al.* 1978; Dahlgren 1981b; Torsvall and Åkerstedt 1987; Torsvall *et al.* 1989). Figure 1 shows a comparison of sleepiness levels using the Karolinska Sleepiness Scale (KSS) (Åkerstedt and Gillberg 1990). For nuclear-power station technicians, maintenance work inside nuclear reactors show a steep increase in sleepiness during the night shift, from 3 ('alert') to 7 ('sleepy, but not fighting sleep') (Kecklund and Åkerstedt 1993b). Long-haul truck drivers increase in a similar way (Kecklund and Åkerstedt 1993a) and air crew on westward (Stockholm–Los Angeles) trans-meridian flights even exceed 7 and finish close to 9 ('very sleepy, fighting sleep, an effort to stay awake') towards the end of the return flight (Lowden and Åkerstedt 1995).

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**Figure 1.** Self-rated alertness in process operators during a night and evening shift ( $n = 15$ ), truck drivers during a night and day drive ( $n = 12$ ), and cabin crew ( $n = 35$ ) during a westward flight and return across nine time zones.

Sleep related electroencephalography/electroculography (EEG/EOG)-changes and performance lapses usually begin to appear at level 7 on the KSS and become very pronounced at level 9 (Åkerstedt and Gillberg 1990; Gillberg *et al.* 1994). In quantitative terms the maximum level of subjective sleepiness is associated with twice the normal levels of alpha and theta power density, while the EOG recording contains more than 50% of slow eye movements.

One may also look at sleepiness in terms of accumulated time with severe symptoms of sleepiness. Thus, KSS-ratings around 8–9 correspond to 40 min of rated 'heavy eye-lids' or 8 min of 'difficulties keeping eyes open' during an 8–10 h work period (Gillberg *et al.* 1994). Usually, the relation of subjective sleepiness with performance is a close one, with major performance lapses occurring at the high levels usually encountered in shift work (Gillberg *et al.* 1994).

Interestingly, sleepiness is also evidenced in the officially sanctioned night-shift naps which are seen in some cultures, particularly in Japan (Kogi 1981). It is also reported in western countries, however. Thus, for example, Danish policemen are known to take an 'unofficial' nap when there is little to do during the night shift (Andersen 1970). Anecdotally, this behaviour is also well known in other occupational groups when work load permits it, such as in nursing.

It should be emphasized, however, that not only the night shift is affected by sleepiness. Also, the return to day work is associated with a considerable increase in sleepiness. Furthermore, morning shifts (starting between 04.00 and 07.00 hours) may be perceived as very fatigue inducing (Kecklund *et al.* 1994). In particular, the difficulty rising and the associated inertia may be the most important disadvantage of shift work from the point of view of the shift worker (Åkerstedt *et al.* 1991). The earlier the shift starts, the more sleepiness will be experienced during the day

(Kecklund *et al.* 1994). This effect of early starts may also be seen in pilots on long-haul flights (Gander and Graeber 1987).

## PHYSIOLOGICAL SLEEPINESS

Physiological measures give strong support to the notion of night-shift sleepiness. In an EEG-study of night workers at work (train drivers) we found that one-quarter showed pronounced increases in alpha (8–12 Hz) and theta (4–8 Hz) activity, as well as slow eye movements (SEM) towards the early morning (Torsvall and Åkerstedt 1987). The correlations with ratings of sleepiness were quite high ( $r = 0.74$ ). In some instances obvious performance lapses, such as driving against a red light, occurred during bursts of slow eye movements (SEM) and of alpha/theta activity. The pattern is very similar in truck drivers during long-haul (8–10 h) drives (Kecklund and Åkerstedt 1993a) (Horne and Reyner, this issue pp. 23–29).

Caille and Bassano (1977) demonstrated a strongly increased alpha and theta activity (spectral analysis) towards the end of a task involving driving a car at night. Very similar results from simulated night driving have been presented by Fruhstorfer *et al.* (1977). The latter also demonstrated a parallel increase in the duration of eye blinks and decrease of EOG velocity. Haslam (1982) who studied sleep-deprived soldiers, found an increase of the (visually scored) alpha index during the night hours. Airline pilots also develop similar EEG and EOG changes in connection with night flights (Samel *et al.*, this issue pp. 30–36).

In process operators, not only sleepiness-related increases in alpha and theta activity were found, but also fully-fledged sleep (Torsvall *et al.* 1989). Such incidents of proper sleep occurred in approximately one-quarter of the subjects. Usually they occurred during the second half of the night shift and never in connection with any other shift. Importantly, sleep on the job was not condoned by the company, nor was there any official awareness that sleep would or could occur during work hours. Interestingly, the subjects were unaware of having slept, but were aware of sleepiness.

The occurrence of the night-work sleep intrusions discussed above is comparable to those seen in, for example, narcoleptic patients (Broughton *et al.* 1988), even if the duration of the bouts are shorter and temporally rather restricted. It should also be emphasized that the types of EEG changes described in the shift workers above are clearly related to performance lapses and errors (Daniel 1966; O'Hanlon and Beatty 1977; Torsvall and Åkerstedt 1988b). Thus, high levels of alpha and/or theta power density are virtually incommensurate with perceptual or cognitive performance—the severely sleepy individual is not functioning and perceives major difficulties keeping his eyes open and is, as mentioned, aware of 'fighting sleep' (level 8–9 on the KSS scale) (Åkerstedt and Gillberg 1990).

Incidentally, the amount of alpha power density is usually a direct function of the eye closure duration—long blinks—whereas much of the EEG delta power activity seems to be due to eye-blink artefacts (Torsvall and Åkerstedt 1985).

Furthermore, a number of laboratory studies show that when work/activity is extended into the night, the hours between 04.00 hours and 07.00 hours in the morning will show: a strong increase in alpha and theta activity in ambulatory or sedentary subjects (Åkerstedt and Gillberg 1990), as well as a major decrease (down to 2–4 min) in sleep latency (Carskadon and Dement 1977; Webb 1978; Walsh *et al.* 1986). The latter corresponds to the post-night shift sleep latencies in the field studies cited above.

The levels seen in these studies would be interpreted as pathological sleepiness, should they be observed during a dayshift. They also fall below the levels seen in connection with, for example, moderate intake of alcohol or hypnotics (Roehrs *et al.* 1993).

## SLEEP

Most survey studies have found sleep disturbances to be a major complaint of shiftworkers (Graf *et al.* 1958; Thiis-Evensen 1958; Menzel 1962; Aanonsen 1964; Andersen 1970). EEG studies of sleep in rotating shift workers have shown fairly consistent results, whether recorded in the natural sleep environment (Foret and Lantin 1972; Foret and Benoit 1978; Tilley *et al.* 1981; Torsvall *et al.* 1981; Torsvall *et al.* 1989; Åkerstedt *et al.* 1990; Åkerstedt *et al.* 1991), or in the laboratory (Ehrenstein *et al.* 1970; Foret and Benoit 1974; Matsumoto 1978; Dahlgren 1981a). Thus, day sleep after night work and early night sleep before morning work is 2–4 hours shorter than night sleep. The shortening is primarily taken out of Stage 2 sleep and rapid eye movement (REM) sleep (dream sleep). Stages 3 and 4 or 'delta power density' ('deep sleep'), do not seem to be affected.

Shifts also differ in that the sleep latency is usually shorter (around 5 min) and REM often appears earlier in day sleep after night work than in night sleep after day work. In sleep before the morning shift sleep latency is instead increased. Furthermore, sleep after the night shift is usually spontaneously terminated, whereas sleep before morning work is terminated (rather unpleasantly) by an awakening device (Åkerstedt *et al.* 1991).

With respect to time for adjustment, Foret and Benoit (1978) found that neither the total sleep length, nor the amount of Stages 3 and 4, recovered significantly over four consecutive nightshifts (all were strongly reduced on the initial day sleep). Dahlgren (1981a) found that sleep length was reduced to 4.5 h (from 6.0 h) on the first nightshift, but increased again over six consecutive nightshifts to reach a level of 5.7 h. The amount of Stage 2 and REM also increased as the amount of waking decreased. Permanent night workers tend to sleep somewhat less than day workers (Lille 1967; Kripke *et al.* 1971; Bryden and Holdstock 1973;

Dahlgren 1981a; Tepas *et al.* 1981; Tepas and Mahan 1989). The latter showed that the first day sleep was reduced by 1.1 h (compared to normal night sleep) and actually decreased a further 0.8 h over the six nightshifts.

To work 'on-call' is a rather special form of shift work, but may have similar effects. Thus, it has been demonstrated that physicians in smaller hospitals lost about 50% of total sleep time (TST) (mainly Stages 2 and REM) (Åkerstedt *et al.* 1990). Merchant marine engineers showed reduced TST and slow-wave activity while sleeping on-call—but also during nights when no calls to work occurred (Torsvall and Åkerstedt 1988a). This was interpreted as an effect of increased tension due to the 50% risk of being called to work. Both physicians and machine officers showed increased sleepiness during work.

## THE CIRCADIAN AND HOMEOSTATIC MECHANISM

The reason for the reduced alertness associated with irregular work hours is that displaced hours of work are in conflict with basic biological principles regulating the timing of rest and activity, that is the circadian and homeostatic regulatory systems. The effect involves shortened sleep, an unfavourable time of day for work, and an extended duration of wakefulness.

The main cause of shift-work sleepiness may be the circadian rhythm. A number of studies involving normal night-time sleep (Folkard and Monk 1985), sleep deprivation (Fröberg *et al.* 1975), spontaneous desynchronization (Wever 1979; Czeisler *et al.* 1980), and forced desynchronization (Folkard *et al.* 1985; Dijk *et al.* 1992) have shown that alertness and performance exhibit a time of day pattern with a maximum in the late afternoon and a trough in the early morning around 05.00 hours. The oscillation is clearly a self-sustained circadian rhythm, driven by the pacemaker in the suprachiasmatic nuclei of the hypothalamus (Moore and Eichler 1972; Klein *et al.* 1991). The circadian characteristics have been described for a large number of circadian functions. Of those relevant in the present context, body temperature has a maximum around 17.00 hours and a minimum around 05.00 hours (Aschoff 1965; Czeisler *et al.* 1980; Minors and Waterhouse 1984), melatonin has a maximum around 04.00 hours in the morning and a minimum around 16.00 hours and seems closely related to temperature and to alertness (Åkerstedt *et al.* 1979) (Fig. 2). The temperature-melatonin interaction has recently been described by Cagnacci (1993). Clearly work at the circadian nadir will be carried out at low levels of physiological activation, subjective alertness, or behavioural efficiency (Fig. 2). The latter two variables also exhibit a superimposition due to a homeostatic fall of alertness (time awake) as discussed below.

Studies of temporal isolation and forced desynchronization have demonstrated that the circadian pacemaker will free-run at a non-24 h frequency (Aschoff 1965; Wever 1969;

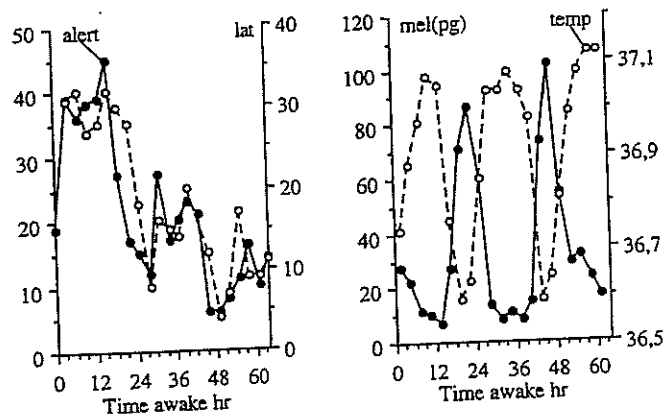


Figure 2. Results from 64 h of sleep loss ( $n = 12$ ). To the left subjective alertness and time elapsed (latency = 'lat') to the first performance miss on a vigilance test. Right: Urinary excretion of melatonin and rectal temperature. Awakening at 07.00 hours.

Folkard *et al.* 1985) and it has been demonstrated that the adjustment to a new circadian-phase position will occur at the speed of about 1 h per day (Mills 1976; Wever 1980). The mechanism seems to be light exposure at a particular circadian phase (Czeisler *et al.* 1981; Czeisler *et al.* 1986; Minors *et al.* 1991; Czeisler and Dijk, this issue pp. 70–73). The circadian adjustment of shift workers, however, is counteracted by a light pattern in opposition to night-work hours. Thus, it appears, that only very marginal circadian adjustment occurs in shift workers (Åkerstedt 1985) (Knauth, this issue pp. 41–46). Several papers have recently shown that light exposure timed to the hours before the circadian trough may remove most of the sleepiness during night work (or rather 'activity') and improve subsequent sleep (Czeisler *et al.* 1990; Eastman 1992; Czeisler 1994; Eastman *et al.* 1994).

Shortened sleep has long been an obvious suspect in shift-work sleepiness because of the 2–4 h reduction in sleep length in connection with night and morning shifts. There are, however, no experimental data on the effects of different amounts of sleep in shift work, although more indirect data are available, for example, an increased tendency to nap in individuals with decreased night- or morning-shift sleep (Åkerstedt and Torsvall 1985). As discussed by Gillberg, this issue pp. 37–40, a 2–4 h of reduction of night sleep will reduce alertness, although probably not enough to make it the major cause of night-shift sleepiness—the recuperative value of the last 2–3 h of an 8 h sleep seems quite low.

The reason for the shortened daytime sleep has frequently been sought in environmental disturbances (Thiis-Evensen 1958; Menzel 1962; Ehrenstein *et al.* 1970; Knauth and Rutenfranz 1972; Knauth and Rutenfranz 1975). However, sleep after a night awake is also shortened under optimal laboratory conditions (Åkerstedt and Gillberg 1981). The reason for the truncated morning sleep after night work is instead a strong time-of-day effect (Åkerstedt and Gillberg 1981). As bedtime is delayed from conventional hours, sleep

length will fall to about 4.5 h for morning to noon bedtimes, and then recover towards the subsequent evening (Fig. 3). The effect is obviously circadian and closely related to the body-temperature cycle (Czeisler *et al.* 1980). Thus, sleep is very difficult at the acrophase (maximum) of the body-temperature rhythm and very easy at the nadir (minimum). Dijk *et al.* (1994) have recently suggested that the circadian rhythm of sleep propensity serves to consolidate sleep and wakefulness. One should keep in mind though, that sleep length is equally determined by prior wakefulness. Thus, a 5-h sleep at noon after a night shift would shrink to 3.5 h if a 2 h nap was permitted during the night, or to 2 h if a full night sleep (7–8 h) was permitted (Åkerstedt and Gillberg 1986).

The mechanism behind the short sleep before the morning shift is the need to terminate sleep very early in the morning without the individual being able to advance bedtime to compensate fully for this. The latter failure may be partly social but there is also a strong circadian influence on sleep latency, making early initiation of sleep very difficult (Åkerstedt *et al.* 1992; Folkard and Barton 1993). This early evening time of sleep resistance has been called a 'forbidden zone' for sleep (Lavie 1986).

A sleep shortened through truncation will clearly increase the time spent awake. This factor, which is also increased by delaying work hours, is less established in shift work, but early experimental data from sleep-deprivation studies clearly show a pronounced fall of alertness and performance across time, levelling out towards days 3–4 (controlling for circadian influence) (Williams *et al.* 1959; Fröberg *et al.* 1975). Figure 2 illustrates the steep fall of alertness and performance during 64 h of sleep deprivation, together with the superimposition of a circadian component (Gillberg and Åkerstedt 1981; Åkerstedt *et al.* 1982). Recent studies have found the same phenomenon of gradual fall in alertness/performance in connection with forced desynchronization (Folkard and Åkerstedt 1991; Dijk *et al.* 1992).

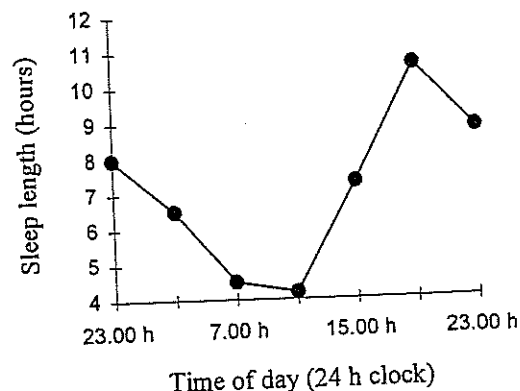


Figure 3. Total sleep time (TST) when sleep is displaced to different times of day and with 16–40 h of prior time awake ( $n = 8$ ).

The effects of prior time awake should be viewed against the fact that the night shift starts 10–16 h after rising, in contrast to the 1–2 h of the morning shift, or the 4–6 h of the afternoon shift. Thus, night work is usually preceded by an extended period of time awake, compared to the morning and evening work periods. Similarly, an early start of morning work will involve a longer period of wakefulness than a late start and this will increase afternoon sleepiness (Kecklund *et al.* 1994).

Finally, whereas the timing of work and sleep will be the major determinant of sleepiness in shift work, an important additional factor will be external stimulation. The central nervous system is built to respond to change and any situation that creates monotony (absence of stimuli, sequences of similar stimuli, etc.) will impair alertness (Wilkinson 1964; Wilkinson 1969; Kjellberg 1977). The monotony effects in real life work are likely to be somewhat different, however, since the individual will try to counteract them. As yet, however, we have very little information about the effects on safety and alertness of 'monotony' in real life situations.

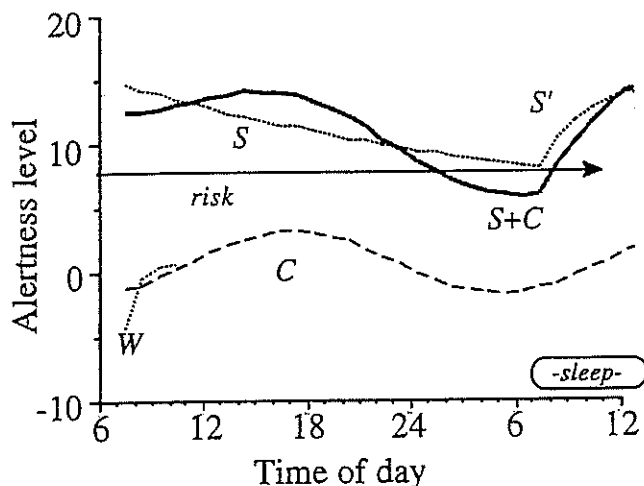
## A MODEL

In order to illustrate, explain and predict variations in alertness related to irregular sleep/wake patterns, we have constructed a quantitative model that uses sleep/wake timing to predict alertness (Folkard and Åkerstedt 1991). The work was inspired by the 'two-process model of sleep

regulation', which had shown that sleep length and slow wave activity could be described by a combination of a homeostatic (prior time awake or amount of prior sleep) and a circadian influence (Borbély 1982). Using subjective alertness data from a number of experiments of altered sleep/wake patterns it was also found that alertness was indeed predictable from a circadian and a homeostatic component—plus a component for sleep inertia. The output of the model has been validated against subjective ratings, performance and EOG measures of sleepiness, and has shown a considerable accuracy (Folkard and Åkerstedt 1991; Åkerstedt and Folkard 1995).

Modelling empirical data we found that alertness was predictable from three parameters: S, C, and W (Fig. 4). Process C represents sleepiness due to circadian influences and has a sinusoidal form with an afternoon peak (Fig. 4). Process S is an exponential function of the time since awakening, is high on awakening, falls rapidly initially and gradually approaches a lower asymptote. At sleep onset process S is reversed and called S' and recovery occurs in an exponential fashion that initially increases very rapidly but subsequently levels off towards an upper asymptote. Total recovery is usually accomplished in 8 h. The final component is the wakeup Process W, or sleep inertia, after forced awakenings. This function is also exponential but with an even steeper initial decrease, i.e. after the first hour most of the inertia has already dissipated. W is subtracted from the S + C level. The model also accounts for sleep latency and sleep length (Åkerstedt and Folkard, *in press*, a&b).

The predicted alertness is expressed as the arithmetic sum of the two functions above. The scale of the model ranges normally from 1–16, but in practice '3' corresponds to extreme sleepiness and '14' to high alertness and '7' to a threshold below which EEG/EOG sleep intrusions will appear within 5 min under conditions of low external stimulation (Folkard and Åkerstedt 1991). In Fig. 4, S + C shows predicted alertness when wakefulness is extended by 8 h (to 24 h), as is frequently the case, for example, with a first night shift. This particular prediction assumes that awakening occurs at 07.00 hours in the morning after an 8 h sleep, and thereafter no sleep occurs (due to the night shift) until 07.00 hours the following day. The combined effect of S + C (long time awake and the circadian downswing) yields a fall of alertness during the night, with a trough in the early morning. After sleep is started, the steep recovery of factor S, together with the circadian upswing causes a rapid increase in (latent) alertness during sleep.



**Figure 4.** Parameters of the three-process model of alertness regulation. The figure illustrates the 36-h period of a first night shift—awakening in the morning and continuous wakefulness until bedtime at 07.00 hours the next morning and the subsequent morning sleep. S = homeostatic component during waking; S' = homeostatic component during sleep; C = circadian component. W = wake up component (very negative at wake-up and approaching zero); S + C = the alertness prediction (excluding W). Level of risk = '7'.

## CONCLUSIONS

Subjective, behavioural, and physiological sleepiness clearly occurs in abundance in connection with night work, and also morning work to some extent. It afflicts virtually all individuals almost without exception and will reach severe performance incapacitating levels in a majority of subjects

and outright incidents of sleep in a large minority. Clearly, the level of sleepiness is sufficient to be associated with sleep intrusions in the EEG and with an associated cessation of behaviour. The latter will, if coincident with safety-related performance demands, be associated with increased risk of accidents.

The alertness deficit is caused by the displacement of work to the circadian phase which is least conducive to alert behaviour, by extension of the time spent awake, and by the reduction of sleep length (due to circadian interference with sleep). Sleepiness will be extreme when the three causes are simultaneously operative.

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